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NEWS 1		Web Page URLs for STN Seminar Schedule - N. America
NEWS 2	Dec 17	The CA Lexicon available in the CAPLUS and CA files
NEWS 3	Feb 06	Engineering Information Encompass files have new names
NEWS 4	Feb 16	TOXLINE no longer being updated
NEWS 5	Apr 23	Search Derwent WPINDEX by chemical structure
NEWS 6	Apr 23	PRE-1967 REFERENCES NOW SEARCHABLE IN CAPLUS AND CA
NEWS 7	May 07	DGENE Reload
NEWS 8	Jun 20	Published patent applications (A1) are now in USPATFULL
NEWS 9	JUL 13	New SDI alert frequency now available in Derwent's DWPI and DPCI
NEWS 10	Aug 23	In-process records and more frequent updates now in MEDLINE
NEWS 11	Aug 23	PAGE IMAGES FOR 1947-1966 RECORDS IN CAPLUS AND CA
NEWS 12	Aug 23	Adis Newsletters (ADISNEWS) now available on STN
NEWS 13	Sep 17	IMSworld Pharmaceutical Company Directory name change to PHARMASEARCH
NEWS 14	Oct 09	Korean abstracts now included in Derwent World Patents Index
NEWS 15	Oct 09	Number of Derwent World Patents Index updates increased
NEWS 16	Oct 15	Calculated properties now in the REGISTRY/ZREGISTRY File
NEWS 17	Oct 22	Over 1 million reactions added to CASREACT
NEWS 18	Oct 22	DGENE GETSIM has been improved
NEWS 19	Oct 29	AAASD no longer available
NEWS 20	Nov 19	New Search Capabilities USPATFULL and USPAT2
NEWS 21	Nov 19	TOXCENTER(SM) - new toxicology file now available on STN
NEWS 22	Nov 29	COPPERLIT now available on STN
NEWS 23	Nov 29	DWPI revisions to NTIS and US Provisional Numbers
NEWS 24	Nov 30	Files VETU and VETB to have open access
NEWS 25	Dec 10	WPINDEX/WPIDS/WPIX New and Revised Manual Codes for 2002
NEWS 26	Dec 10	DGENE BLAST Homology Search
NEWS 27	Dec 17	WELDASEARCH now available on STN
NEWS 28	Dec 17	STANDARDS now available on STN
NEWS 29	Dec 17	New fields for DPCI
NEWS 30	Dec 19	CAS Roles modified
NEWS 31	Dec 19	1907-1946 data and page images added to CA and CAplus

NEWS EXPRESS	August 15 CURRENT WINDOWS VERSION IS V6.0c, CURRENT MACINTOSH VERSION IS V6.0 (ENG) AND V6.0J (JP), AND CURRENT DISCOVER FILE IS DATED 07 AUGUST 2001
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FILE 'HOME' ENTERED AT 10:41:46 ON 03 JAN 2002

=> File medline embase biosis cancerlit
COST IN U.S. DOLLARS

	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.30	0.30

FILE 'MEDLINE' ENTERED AT 10:43:07 ON 03 JAN 2002

FILE 'EMBASE' ENTERED AT 10:43:07 ON 03 JAN 2002

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FILE 'CANCERLIT' ENTERED AT 10:43:07 ON 03 JAN 2002

=> S (nude or SCID or immunodeficient) same (rodent or mouse)
MISSING OPERATOR EFICIENT) SAME
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nested terms that are not separated by a logical operator.

=> S (nude or SCID or immunodeficient)same(rodent or mouse)
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nested terms that are not separated by a logical operator.

=> S (nude or SCID or immunodeficient)with(rodent or mouse)
MISSING OPERATOR DEFICIENT)WITH
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nested terms that are not separated by a logical operator.

=> S (nude or SCID or immunodeficient) with (rodent or mouse)
MISSING OPERATOR EFICIENT) WITH
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> S (nude or SCID or immunodeficient) and (rodent or mouse)
L1 99460 (NUDE OR SCID OR IMMUNODEFICIENT) AND (RODENT OR MOUSE)

=> S (tumor cells) and (peripheral or Intravenous)
L2 23743 (TUMOR CELLS) AND (PERIPHERAL OR INTRAVENOUS)

=> S (bone metast? or metast?)
L3 602380 (BONE METAST? OR METAST?)

=> S L1 and L2
L4 1643 L1 AND L2

=> S L4 and L3
L5 489 L4 AND L3

=> S L4 same L3
MISSING OPERATOR L4 SAME
The search profile that was entered contains terms or

nested terms that are not separated by a logical operator.

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=> S "L4" same "L3"
L6            0 "L4" SAME "L3"

=> S 'L4' same 'L3'
L7            0 'L4' SAME 'L3'

=> S (L4) same (L3)
MISSING OPERATOR L4) SAME
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nested terms that are not separated by a logical operator.

=> L5 and (anti-IL-2 receptor antibody)
L5 IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> S L5 and (anti-IL-2 receptor antibody)
L8            0 L5 AND (ANTI-IL-2 RECEPTOR ANTIBODY)

=> S L5 and (NK cells)
L9           23 L5 AND (NK CELLS)

=> D his

     (FILE 'HOME' ENTERED AT 10:41:46 ON 03 JAN 2002)

     FILE 'MEDLINE, EMBASE, BIOSIS, CANCERLIT' ENTERED AT 10:43:07 ON 03 JAN
     2002
L1         99460 S (NUDE OR SCID OR IMMUNODEFICIENT) AND (RODENT OR MOUSE)
L2         23743 S (TUMOR CELLS) AND (PERIPHERAL OR INTRAVENOUS)
L3        602380 S (BONE METAST? OR METAST?)
L4          1643 S L1 AND L2
L5           489 S L4 AND L3
L6             0 S "L4" SAME "L3"
L7             0 S 'L4' SAME 'L3'
L8             0 S L5 AND (ANTI-IL-2 RECEPTOR ANTIBODY)
L9            23 S L5 AND (NK CELLS)

=> Dup rem L9
PROCESSING COMPLETED FOR L9
L10           10 DUP REM L9 (13 DUPLICATES REMOVED)

=> Dup rem L5
PROCESSING COMPLETED FOR L5
L11          226 DUP REM L5 (263 DUPLICATES REMOVED)

=> Display L10 IBIB ABS TOTAL

L10  ANSWER 1 OF 10     MEDLINE                          DUPLICATE 1
ACCESSION NUMBER:    2000242958     MEDLINE
DOCUMENT NUMBER:     20242958   PubMed ID: 10782864
TITLE:               Immunotherapy with vaccines combining MHC class II/CD80+
                     tumor cells with interleukin-12 reduces
                     established metastatic disease and stimulates
                     immune effectors and monokine induced by interferon gamma.
AUTHOR:              Pulaski B A; Clements V K; Pipeling M R; Ostrand-Rosenberg
                     S
CORPORATE SOURCE:    Department of Biological Sciences, University of Maryland,
                     Baltimore 21250, USA.
```

CONTRACT NUMBER: R01CA52527 (NCI)
SOURCE: CANCER IMMUNOLOGY, IMMUNOTHERAPY, (2000 Apr) 49 (1) 34-45.
Journal code: CN3; 8605732. ISSN: 0340-7004.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200005
ENTRY DATE: Entered STN: 20000518
Last Updated on STN: 20000518
Entered Medline: 20000509

AB Because they are difficult to treat, animal models of widespread, established **metastatic** cancer are rarely used to test novel immunotherapies. Two such **mouse** models are used in this report to demonstrate the therapeutic efficacy and to probe the mechanisms of a novel combination immunotherapy consisting of the cytokine interleukin-12 (IL-12) combined with a previously described vaccine based on MHC class II, CD80-expressing cells. BALB/c **mice** with 3-week established primary 4T1 mammary carcinomas up to 6 mm in diameter and with extensive, spontaneous lung **metastases** show a significant reduction in lung **metastases** following a 3-week course of immunotherapy consisting of weekly injections of the cell-based vaccine plus injections of IL-12 three times per week. C57BL/6 **mice** with 7-day established **intravenous** B16.melF10 lung **metastases** show a similar response following immunotherapy with IL-12 plus a vaccine based on B16 MHC class II, CD80-expressing cells. In both systems the combination therapy of cells plus IL-12 is more effective than IL-12 or the cellular vaccine alone, although, in the 4T1 system, optimal activity does not require MHC class II and CD80 expression in the vaccine cells. The cell-based vaccines were originally designed to activate tumor-specific CD4+ T lymphocytes specifically and thereby provide helper activity to tumor-cytotoxic CD8+ T cells, and IL-12 was added to the therapy to facilitate T helper type 1 lymphocyte (Th1) differentiation. In vivo depletion experiments for CD4+ and CD8+ T cells and natural killer (**NK**) **cells** and tumor challenge experiments in beige/ **nude**/XID **immunodeficient mice** demonstrate that the therapeutic effect is not exclusively dependent on a single cell population, suggesting that T and **NK cells** are acting together to optimize the response. IL-12 may also be enhancing the immunotherapy via induction of the chemokine Mig (monokine induced by interferon gamma), because reverse PCR experiments demonstrate that Mig is present in the lungs of **mice** receiving therapy and is most likely synthesized by the **tumor cells**. These results demonstrate that the combination therapy of systemic IL-12 and a cell-based vaccine is an effective agent for the treatment of advanced, disseminated **metastatic** cancers in experimental **mouse** models and that multiple effector cell populations and anti-angiostatic factors are likely to mediate the effect.

L10 ANSWER 2 OF 10 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1999192615 MEDLINE
DOCUMENT NUMBER: 99192615 PubMed ID: 10090831
TITLE: Early events of hepatic **metastasis** formation in **mice**: role of Kupffer and **NK-cells** in natural and interferon-gamma-stimulated defense.
AUTHOR: Rushfeldt C; Sveinbjornsson B; Seljelid R; Smedsrod B
CORPORATE SOURCE: Department of Experimental Pathology, Institute of Medical Biology, Tromso, N-9037, Norway.
SOURCE: JOURNAL OF SURGICAL RESEARCH, (1999 Apr) 82 (2) 209-15.
Journal code: K7B; 0376340. ISSN: 0022-4804.
PUB. COUNTRY: United States

Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200008
ENTRY DATE: Entered STN: 20000811
Last Updated on STN: 20000811
Entered Medline: 20000801

AB Surgical manipulation of a tumor may result in increased influx of **tumor cells** into the systemic and portal circulation and give rise to formation of **metastases**. In addition, major surgery has been reported to cause profound immunosuppression. In an attempt to increase the host-antitumor immune mechanisms following surgery we have studied the effect of preoperative administration of interferon-gamma, related to the antimetastatic effects of Kupffer cells (KC) and natural killer cells (**NK-cells**) in the early phase of liver **metastasis** formation. Colon carcinoma cells were injected into the superior mesenteric vein of syngeneic **mice** and after 17 days **metastases** were quantified by weight, number, and uptake of [125I]iododeoxyuridine. Unstimulated control **mice** developed 10.5 surface nodules per liver 17 days following injection of colon carcinoma cells into the superior mesenteric vein of syngeneic **mice**. This figure was only 2.6 in **mice** stimulated with a single dose of 1000 IU IFN-gamma 4 h prior to inoculation of **tumor cells**. Administration of GdCl3, which is reported to deplete and block the function of Kupffer cells, 24 h prior to tumor cell inoculation resulted in a 5-fold tumor mass increase relative to control. Injection of anti-asiolo-GM1 antiserum, which eliminates the hepatic **NK-cells**, induced a 10-fold increase in tumor mass. These results indicate an important early antimetastatic function of hepatic **NK-cells** and KC and that presurgical administration of IFN-gamma may be important for eliminating circulating **tumor cells** and inhibiting development of residual tumors.

L10 ANSWER 3 OF 10 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 97303203 MEDLINE
DOCUMENT NUMBER: 97303203 PubMed ID: 9159146
TITLE: A lymphocyte-activating monoclonal antibody induces regression of human tumors in severe combined **immunodeficient mice**.
AUTHOR: Hardy B; Kovjazin R; Raiter A; Ganor N; Novogrodsky A
CORPORATE SOURCE: Felsenstein Medical Research Center, Rabin Medical Center, Belinson Campus, Petah Tikva 49100, Israel.
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1997 May 27) 94 (11) 5756-60. Journal code: PV3; 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199706
ENTRY DATE: Entered STN: 19970630
Last Updated on STN: 19970630
Entered Medline: 19970619

AB Monoclonal antibodies were raised against Daudi B-lymphoblastoid cell line membranes. An mAb (BAT) was selected for its ability to stimulate human and murine lymphocyte proliferation. BAT induced cytotoxicity in human and murine lymphocytes against natural killer cell-sensitive and -resistant tumor cell lines. A single **intravenous** administration of BAT to **mice** that had been inoculated with various murine tumors (e.g., B16 melanoma, 3LL carcinoma, and methylcholanthrene fibrosarcoma) resulted in striking antitumor effects as manifested by complete tumor regression

and prolonged survival of the treated **mice**. BAT exhibited a diminished but significant antitumor effect in athymic **nude mice**, which are deficient in T lymphocytes, and in beige **mice**, which are deficient in **NK cells**. Furthermore, selective depletion of T or **NK cells** in **mice** reduced the response to the antitumor effect of BAT. These data indicate a dual role for T and **NK cells** in mediating the antitumor activity of BAT. We report here on the antitumor activity of BAT mAb on human tumor xenografts in **mice**. BAT demonstrated an antitumor effect in **nude mice** bearing human colon carcinoma (HT29) xenografts. It failed, however, to inhibit established lung **metastases** in severe combined **immunodeficient** (**SCID**) **mice** that had been inoculated (i.v.) with SK28 human melanoma. Engraftment of human lymphocytes into **SCID mice** bearing human melanoma xenografts rendered them responsive to the antitumor effect of BAT. The efficacy of BAT in the regression of human tumors by activation of human lymphocytes indicates its potential clinical use.

L10 ANSWER 4 OF 10 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 96427371 MEDLINE
DOCUMENT NUMBER: 96427371 PubMed ID: 8830735
TITLE: Introduction of the interferon gamma gene into **mouse** T lymphoma cells with low MHC class I-expression results in selective induction of H-2Dk and concomitant enhanced **metastasis**.
AUTHOR: Geldhof A B; VandenDriessche T; Opdenakker G; De Baetselier P
CORPORATE SOURCE: Laboratory of Cellular Immunology, Flemish Interuniversity Institute for Biotechnology, Free University of Brussels, Belgium.
SOURCE: CANCER IMMUNOLOGY, IMMUNOTHERAPY, (1996 Jul) 42 (6) 329-38. Journal code: CN3; 8605732. ISSN: 0340-7004.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199611
ENTRY DATE: Entered STN: 19961219
Last Updated on STN: 19961219
Entered Medline: 19961104

AB Interferon-gamma (IFN gamma)-induced up-regulation of MHC class I expression on **tumor cells** can induce a potent CD8-mediated antitumor response. Consequently, many investigators have proposed IFN gamma gene transfection as a means to immunogenize **tumor cells** and to vaccinate against **metastatic** disease. In this study, we demonstrate that transfection of the IFN gamma gene in a BW5147 variant (LiDlo) with low MHC class I expression results in a selective induction of H-2Dk but unaltered H-2Kk expression. In earlier reports we demonstrated a positive correlation between H-2Dk expression and enhanced **metastatic** potential of BW variants. In accordance with these observations, we observed that **intravenous** inoculation of LiDlo(IFN gamma) variants into syngeneic AKR **mice** led to enhanced **metastasis** as compared to parental LiDlo and LiDlo(neo) control transfectants. **Tumor cells**, derived from local subcutaneous tumors or sporadic **metastases** from **mice** inoculated with LiDlo **tumor cells**, were found to up-regulate H-2Dk selectively. Anti-asialoGM1 treatment of AKR **mice** allowed rapid experimental **metastasis** formation by the LiDlo and LiDlo(neo) variants, indicating that natural killer (**NK**) **cells** control the **metastatic** behavior of these **tumor cells**. This was corroborated by in vitro

cytotoxicity experiments, demonstrating the LiDlo and LiDlo(neo) **tumor cells** were NK-sensitive, while the BW IFN gamma transfectants became resistant to lymphokine-activated killer cells and poly(I).poly(C)-induced **NK cells**. We thus conclude that (a) IFN gamma up-regulates selectively the MHC class I antigen H-2Dk, (b) H-2Dk governs susceptibility towards **NK cells**, and (c) NK susceptibility determines the experimental **metastatic** behavior of BW **tumor cells**.

L10 ANSWER 5 OF 10 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 96332590 MEDLINE
DOCUMENT NUMBER: 96332590 PubMed ID: 8760590
TITLE: Novel **metastasis** model of human lung cancer in **SCID mice** depleted of **NK cells**.
AUTHOR: Yano S; Nishioka Y; Izumi K; Tsuruo T; Tanaka T; Miyasaka M; Sone S
CORPORATE SOURCE: Third Department of Internal Medicine, University of Tokushima School of Medicine, Japan.
SOURCE: INTERNATIONAL JOURNAL OF CANCER, (1996 Jul 17) 67 (2) 211-7.
Journal code: GQU; 0042124. ISSN: 0020-7136.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199609
ENTRY DATE: Entered STN: 19960924
Last Updated on STN: 19970203
Entered Medline: 19960916

applicants own

AB **Metastasis** is a critical problem in the treatment of human lung cancer. Thus, a suitable animal model of **metastasis** of human lung cancer is required for in vivo biological and preclinical studies. In this study, we tried to establish a suitable model for this, using **SCID mice**. Neither human SCLC H69/VP cells (5 x 10(6)) nor squamous-cell carcinoma RERF-LC-AI cells (1 x 10(6)), injected through a tail vein, formed **metastases** in untreated **SCID mice**. Pre-treatment of **SCID mice** with anti-asialo GM1 serum resulted in only a few **metastases** of H69/VP cells, but pre-treatment with anti-**mouse** IL-2 receptor beta chain Ab (TM-beta 1) resulted in numerous lymph-node **metastases** 56 days after tumor inoculation. H69/VP-M cells, an in vivo-selected variant line, formed significant numbers of lymph-node **metastases** even in **SCID mice** pre-treated with anti-asialo GM1 serum. **SCID mice** depleted of **NK cells** by treatment with TM-beta 1 showed different patterns of **metastasis** when inoculated intravenously with the 2 different human lung cancer cell lines (H69/VP and RERF-LC-AI cells): H69/VP cells formed **metastases** mainly in systemic lymph nodes and the liver, whereas RERF-LC-AI cells formed **metastases** mainly in the liver and kidneys, with only a few in lymph nodes. A histopathological study showed that the **metastatic** colonies consisted of cancer cells. The numbers of **metastatic** colonies formed by the 2 cell lines increased with the number of cells inoculated. TM-beta 1 treatment of **SCID mice** efficiently removed **NK cells** from **peripheral** blood for at least 6 weeks, whereas, after treatment of the **mice** with anti-asialo GM1 serum, **NK cells** were recovered within 9 days. These findings suggest that NK-cell-depleted **SCID mice** may be useful as a model in biological and pre-clinical studies on **metastasis** of human lung cancer.

L10 ANSWER 6 OF 10 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 96400411 MEDLINE
DOCUMENT NUMBER: 96400411 PubMed ID: 8806787
TITLE: Role of natural killer cells on engraftment of human lymphoid cells and on **metastasis** of human T-lymphoblastoid leukemia cells in C57BL/6J-**scid** **mice** and in C57BL/6J-**scid** bg **mice**
.
AUTHOR: Christianson S W; Greiner D L; Schweitzer I B; Gott B; Beamer G L; Schweitzer P A; Hesselton R M; Shultz L D
CORPORATE SOURCE: Jackson Laboratory, Bar Harbor, Maine.
CONTRACT NUMBER: AI 30389 (NIAID)
CA 20408 (NCI)
CA34196 (NCI)
SOURCE: CELLULAR IMMUNOLOGY, (1996 Aug 1) 171 (2) 186-99.
Journal code: CQ9; 1246405. ISSN: 0008-8749.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199611
ENTRY DATE: Entered STN: 19961219
Last Updated on STN: 19961219
Entered Medline: 19961101

AB The severe combined immunodeficiency (**scid**) mutation was backcrossed onto the C57BL/6J strain background in order to study the role of natural killer (**NK**) **cells** in rejection of normal and malignant human lymphohematopoietic cells. C57BL/6J-**scid**/**scid** **mice** showed severe loss of mature T and B cells accompanied by increased percentages of NK1.1+ cells and myeloid cells. Although little or no serum immunoglobulin was detectable prior to 2 months of age, all **mice** tested had circulating immunoglobulin by 7.5 months of age. C57BL/6J-**scid**/**scid** **mice** had markedly elevated levels of both hemolytic complement activity and NK cell activity compared with C57BL/6J - (+/+) controls. Weekly injections with anti-NK1.1 antibody resulted in elimination of NK cell activity in C57BL/6J-**scid**/**scid** **mice** throughout 8 weeks of treatment. Although human CEM-C7 T lymphoblastoid **tumor** **cells** grew slowly in unmanipulated C57BL/6J-**scid**/**scid** **mice**, anti-NK1.1 treatment resulted in increased growth accompanied by **metastasis** of human lymphoma cells to the brain, liver, and kidney. In contrast to T lymphoblastoid **tumor** **cells**, nonmalignant human **peripheral** blood mononuclear cells engrafted at low levels in anti-NK1.1-treated as well as in unmanipulated C57BL/6-**scid**/**scid** **mice**. Backcrossing of the beige (bgJ) mutation onto the C57BL/6-**scid**/**scid** genetic stock caused decreased NK cell activity accompanied by granulocyte defects. C57BL/6-**scid**/**scid** bgJ)/bgJ) **mice** showed **metastasis** of human CEM-C7 cells to the brain and other organs but supported only low levels of engraftment with human **peripheral** blood mononuclear cells. These results demonstrate that **NK** **cells**, in the absence of an adaptive immune system, function in resistance to **metastasis** of human lymphomas and suggest that innate immune factors in addition to NK cell function mediate resistance to engraftment of normal human **peripheral** blood leukocytes.

L10 ANSWER 7 OF 10 CANCERLIT
ACCESSION NUMBER: 96602133 CANCERLIT
DOCUMENT NUMBER: 96602133
TITLE: Effect of recombinant human interleukin 10 on tumor **metastasis** (Meeting abstract).

AUTHOR: Garaud F; Maxwell E; Li Z; Chen P; Catino J; King I; Zheng L M
CORPORATE SOURCE: Tumor Biology Dpt, Schering-Plough Research Institute, 2015 Galloping Hill Road, Kenilworth, NJ 07033.
SOURCE: Proc Annu Meet Am Assoc Cancer Res, (1995). Vol. 14, pp. A2779.
ISSN: 0197-016X.
DOCUMENT TYPE: (MEETING ABSTRACTS)
FILE SEGMENT: ICDB
LANGUAGE: English
ENTRY MONTH: 199604

AB Human interleukin 10 (hIL-10) inhibits macrophage function as well as proinflammatory cytokines synthesis and enhances IL-2 mediated cell mediated cytotoxicity. We studied the effect of hIL-10 on tumor **metastasis** in various murine and human tumor cell lines. Experimental lung **metastases** were obtained after **intravenous** injection of B16-F10 murine melanoma cells in C57/BL6, athymic nu/nu, and beige **mice**. Spontaneous lung **metastases** were obtained by subcutaneous injection of M27 Lewis Lung carcinoma cells in C57/BL6 and Lox human melanoma cells in **SCID mice**, respectively. hIL-10 (100 ug/kg) was injected intraperitoneally using various dosing schedules. Daily injection of hIL-10 significantly reduced the number of lung **metastases** in both spontaneous and experimental models. Inhibition ranged from 36.7 to 70.5% for B16-F10, 48 to 90.9% for M27, and 60.9 to 78.7% for Lox, respectively. Administration of hIL-10 for 3 days starting from the same day of tumor inoculation resulted in an inhibition rate comparable to daily administration for 9 days in the experimental **metastasis** B16F10 model (43-58.8%). A similar inhibitory effect was observed in athymic nu/nu **mice** (32-58.1%), but not in beige **mice** (5.3%). No direct cytotoxic effect was observed when **tumor cells** were treated with hIL-10 up to 1 ug/ml in vitro. These results demonstrate that hIL-10 has an inhibitory effect on the growth of lung **metastases** in animals inoculated with B16, M27 and Lox **tumor cells**. Our results also suggest a possible involvement of **NK cells** in the inhibitory effect of IL-10.

L10 ANSWER 8 OF 10 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 94346309 MEDLINE
DOCUMENT NUMBER: 94346309 PubMed ID: 7520663
TITLE: Evidence for nutrient modulation of tumor phenotype: impact of tyrosine and phenylalanine restriction.
AUTHOR: Elstad C A; Meadows G G; Aslakson C J; Starkey J R
CORPORATE SOURCE: Department of Pharmaceutical Sciences, College of Pharmacy, Washington State University, Pullman 99164.
CONTRACT NUMBER: CA42465 (NCI)
SOURCE: ADVANCES IN EXPERIMENTAL MEDICINE AND BIOLOGY, (1994) 354 171-83.
Journal code: 2LU; 0121103. ISSN: 0065-2598.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199409
ENTRY DATE: Entered STN: 19941005
Last Updated on STN: 19970203
Entered Medline: 19940922

AB We have shown that Tyr and Phe restriction suppresses the malignant phenotype of the highly invasive and **metastatic** BL6 variant of B16 murine melanoma. Lung-colonizing abilities of Tyr- and Phe-modulated in vivo and in vitro variants of BL6 are inhibited following

intravenous inoculation into **mice** fed normal diet. Although this antimetastatic effect of Tyr and Phe restriction is most likely not due to differences in attachment to endothelium, our data indicate that major impacts of Tyr and Phe restriction are at the level of the tumor, itself. Modulation of host immune responses, which in turn suppresses **metastasis**, does not appear to contribute significantly to the altered phenotype. Although numbers and function of T cells, mast cells, and **NK cells** are affected by Tyr and Phe restriction, they are not involved in the Tyr- and Phe-mediated suppression of tumor growth, **metastasis**, or angiogenesis. Our data do not rule out the importance of other host factors involved in the Tyr and Phe modulation of tumor phenotype. The outcome of this modulation results most likely from complex Tyr/Phe-tumor-host interactions.

L10 ANSWER 9 OF 10 MEDLINE DUPLICATE 8
ACCESSION NUMBER: 92208105 MEDLINE
DOCUMENT NUMBER: 92208105 PubMed ID: 1804312
TITLE: In situ activation of **mouse** lung macrophages by coadministration of liposomes containing the lipopeptide CGP 31362 and interleukin 2 involves interaction with T lymphocytes and natural killer cells.
AUTHOR: Utsugi T; Dinney C P; Killion J J; Brown D; Fidler I J
CORPORATE SOURCE: Department of Cell Biology, University of Texas M. D. Anderson Cancer Center, Houston 77030.
CONTRACT NUMBER: CA-16672 (NCI)
R35-CA 42107 (NCI)
SOURCE: LYMPHOKINE AND CYTOKINE RESEARCH, (1991 Dec) 10 (6) 487-93.
Journal code: A3G; 9107882. ISSN: 1056-5477.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199205
ENTRY DATE: Entered STN: 19920515
Last Updated on STN: 19970203
Entered Medline: 19920507

AB These studies were undertaken to determine the mechanism for augmented tumoricidal activity of alveolar macrophages (AM) in **mice** injected intravenously with multilamellar liposomes containing a lipopeptide analogue of Gram-negative bacteria cell wall (MLV-CGP 31362) and intraperitoneally with interleukin 2 (IL-2). BALB/c **mice** were injected into the kidney with syngeneic renal carcinoma cells. Ten days later, this kidney was resected, and the **mice** were treated intravenously with MLV-CGP 31362 and/or intraperitoneally with IL-2. Treatment with MLV-CGP 31362 led to a reduction in the number of lung **metastases**, whereas treatment with IL-2 alone did not. The coadministration of **intravenous** liposomes and intraperitoneal IL-2 produced significant eradication of lung **metastases**. MLV-CGP 31362 (iv) and IL-2 (ip) were injected both into control immune-competent and **nude mice** or into **mice** whose natural killer (**NK**) **cells** had been depleted by systemic administration of anti-asialo GM1 antibodies. MLV-CGP 31362 activated tumoricidal properties in AM of all groups of **mice**. The additive tumoricidal activation of AM by IL-2 was associated with its effects on both T cells and **NK cells**.

L10 ANSWER 10 OF 10 MEDLINE DUPLICATE 9
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AUTHOR: Hakim A A

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AB The experiments described in this study examined cell membrane oligosaccharides, malignancy-related cell phenotypes and tumor cell susceptibility to the killing effect of human cytotoxic cells. Short term breast carcinoma (BCa) cell lines were prepared from biopsies obtained from patients at each of the pathological Stages I, II, III and from patients with disseminated liver **metastasis**. Five patients at each stage donated the tissue. To obtain large enough quantities, the cells were cultured as monolayers for a brief period, then transferred to roller bottles using serum-free hormone defined medium. Natural killer (**NK**) **cells**, lymphokine (IL-2)-activated killer (LAK), tumor-infiltrating lymphocytes (TIL) and **peripheral** cytotoxic lymphocytes (CTL) from patients with BCa at PS I were used as the effector cells. Susceptibility of the **tumor cells** to the killing effects of the effector cells was monitored by the well established 4 h 51Cr-release assay technique. Growth factor expression, oncogenicity in athymic female **mice** and colonigenicity in soft agar were used as parameters to monitor breast carcinoma cell malignancy phenotypes. The cell membrane oligosaccharides were determined from the carbohydrate elution profiles from BioGel P-6 columns. The results indicate a correlation between progression of malignancy from PS I to the **metastatic** stage PS IV, and the magnitude of malignancy phenotypes, resistance to the host killer cells and oligosaccharide profile shift to a higher molecular size with increased sialylation and fucosylation of the carbohydrate moieties.